Journal of Basics and Applied Sciences Research (JOBASR)
ISSN print: 3026-9091
Volume 2(1) March 2024
DOI: https://doi.org/10.33003/jobasr-2024-v2i1-33

A Study of a Class Continuous SIR Epidemic Model with History

Olopade I. A. [1]*, Mohammed I. T. [2], Philemon M. E. [1], Akinwumi T. O. [3], Sangoniyi S. O. [4], Adeniran G. A. [5], Ajao S. O. [3], and Adewale S. O. [6]

[1]Department of Mathematics and Statistics, Federal University Wukari, PMB 1020, Wukari, Taraba State, Nigeria.
[2]Department of Mathematics and social sciences, Osun state polytechnic Iree. Nigeria .
[3]Department of Mathematics and Computer Science, Elizade University, Ondo State, Nigeria.
[4]Department of Mathematics and Computing Science Education, Emmanuel Alayande University of Education, Oyo, Oyo State, Nigeria.
[5]Department of Physical Sciences, Chrisland University, P.M.B. 2131, Abeokuta, Ogun State,Nigeria.
[6]Department of Pure and Applied Mathematics LAUTECH Ogbomoso.P.M.B 4000, Ogbomoso Oyo State, Nigeria.
*Corresponding Author Email: isaac.olopade@fuwukari.edu.ng

ABSTRACT

The SIR model, an epidemiological model, divides a population into three compartments: Susceptible (S), Infected (I), and Recovered (R). It is widely used to understand the spread of infectious diseases and predict epidemic outcomes based on factors such as transmission rates and population dynamics. A deterministic epidemic mathematical model to describe the transmission dynamics of an infectious disease was constructed and analyzed by incorporating memory term which provides information on the current and past disease states. The model revealed two key equilibria: a disease-free equilibrium and an endemic equilibrium. The calculated basic reproductive numberR_0, was employed to establish that when $R_0 < 1$, the disease-free equilibrium is locally asymptotically stable, while the endemic equilibrium is locally asymptotically stable when $R_0 > 1$. Additionally, we explored the global stability of these equilibria using Lyapunov functions and Dulac's method, respectively. To validate our analytical findings, we conducted numerical simulations of the model which show the importance of history in the dynamic spread and elimination of disease.

Keywords:
Epidemic Model,
Memory term,
Equilibria,
Reproduction Number,
Sensitivity,
Global Stability.

INTRODUCTION

The SIR model is undoubtedly the most famous mathematical model for the spread of an infectious disease. In 1927, Kermack and McKendrick developed the SIR model, which divides a population into three classes: susceptible, infective, and recovered. This model is fundamental for understanding how infectious diseases spread within populations (Kermack and McKendrick 1927).

Compartmental deterministic models are frequently used to model the transmission of infectious diseases, providing a valuable framework for analyzing and predicting how diseases spread within populations (Brauer and Castillo-Chávez 2001, Capasso 2008, Diekmann et al. 2013).

Cooke, 1979 Developed an SIR epidemic model that incorporates a bilinear incidence rate and introduces a discrete time delay represented as βSI. This model was designed to explore the dynamics of infectious diseases transmitted by vectors such as mosquitoes or rats. The time delay component reflects the period during which the infectious agents mature within the vector before being transmitted to humans. This concept of time delay has gained significant biological importance in epidemic modeling, as highlighted in earlier studies (Cooke 1979, Diekmann et al. 2013).

In 2010, Beretta and Takeuchi conducted a study on discrete SIR epidemic models derived from SIR models with distributed delays. They employed a Lyapunov functional technique to analyze these models and found that the global dynamics of each discrete SIR epidemic model can be fully explained by a single threshold parameter. Importantly, they determined that discrete time delays do not significantly affect the global stability of the endemic equilibrium in the model.

Various variations of the SIR compartmental model have been developed, with some of these outlined in (Adeniran et al., 2022, Adesanya et al., 2016, Adewale et al., 2015a; 2015b, Ajao et al., 2023, Akinwumi et al., 2021, Almuqrin et al., 2021, Alzaid et al., 2021, Beretta et al.,

2010, Foy et al. 2010, Olopade et al. 2017; 2021a; 2021b; 2021c; 2022, Adesola et al., 2024a, Philemon et al., 2023, Ramos et al., 2021, Rao et al., 2019, and Srivastava et al., 2019).

The SIR epidemic model is a fundamental tool for understanding, addressing, and finding solutions to epidemic diseases. It offers a structured framework for analyzing disease transmission and is essential for shaping effective public health responses to minimize the impact of infectious outbreaks on society. Epidemiologists and public health officials heavily rely on the SIR model to predict the course of an epidemic, allowing them to strategically implement control measures and allocate resources efficiently. This includes planning for vaccination programs, quarantine strategies, and treatment protocols, as well as considering the historical context of the disease.

The unique contribution of this research lies in its focus on the "memory term," which provides information about both the current and past states of a disease. This inclusion enhances the model's capacity to consider the historical context, offering a more comprehensive and insightful approach to analyzing epidemics.

The paper is structured as follows:

MATERIALS AND METHODS

In this section, we present the mathematical formulation of an SIR epidemic model. Here, the total population, denoted as$N$, is divided into three distinct classes: susceptible(S), infected(I), and removed (or recovered)(R), individuals. The model assumes that the disease is transmitted from infected individuals to susceptible individuals through direct contact.

In our model, it is assume that the total recruitment at any given time is represented as 'b' and all newly recruited individuals are placed in the susceptible class. We define β as the disease transmission rate, and the non-linear incidence rate is denoted as $\frac{\beta SI}{1+\alpha I}$ where 'α'is the memory term that provides information about the current and past disease states. The population of susceptible individuals is reduced by the natural death rate'μ'_1. Hence;

$$\frac{dS}{dt} = b - \frac{\beta SI}{1+\alpha I} - \mu_1 S \quad (1)$$

The infected population in the model grows as new infections occur through the $\frac{\beta SI}{1+\alpha I}$ simultaneously, the population decreases due to the natural death rate and recovery rates represented by$(\mu_2 \& \theta)$ respectively. Therefore;

$$\frac{dI}{dt} = \frac{\beta SI}{1+\alpha I} - (\mu_2 + \theta)I \quad (2)$$

The population of recovered individuals in the model increases due to the recovery rate $\theta$and decreases as a result of the natural death rateμ_3. Therefore, the time rate of change for the population of recovered individuals can be described by the following equation;

$$\frac{dR}{dt} = \theta I - \mu_3 R \quad (3)$$

All parameters within equations (1) to (3) are nonnegative. These equations can be combined into the following unified form.

$$\begin{aligned} \frac{dS}{dt} &= b - \frac{\beta SI}{1+\alpha I} - \mu_1 S \\ \frac{dI}{dt} &= \frac{\beta SI}{1+\alpha I} - (\mu_2 + \theta)I \\ \frac{dR}{dt} &= \theta I - \mu_3 R \end{aligned} \quad (4)$$

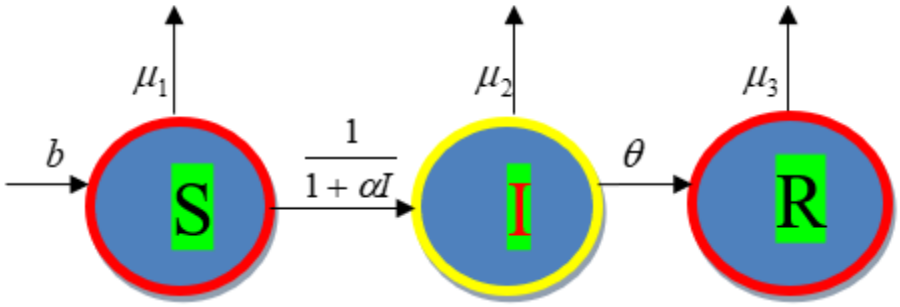

Figure 1. Model flow chart of SIR

The model variables and parameters are defined as follows;

Table 1: Description of Variables

Parameters	Description
$S(t)$	Proportion of the population Susceptible to the disease at time t.
$I(t)$	Proportion of the population of Infective members at time t.
$R(t)$	Proportion of the population who have been removed from the possibility of infection at time t.

Table 2: Description of Parameters

Parameters	Description	Value	Source
μ_1	Death rates of the susceptible class	0.02	Safiel et al. 2012
μ_2	Death rates of the infectious class	0.02	Ibrahim et al. 2015
μ_3	Death rates of the removed class	0.02	Ibrahim et al. 2015
b	Recruitment rate of the population	5	Calibrated
β	Contact rate	0.001	Sajid et al. 2013
α	Memory term	0.01	Calibrated
θ	Treatment rate	0.1	Sajid et al. 2013

Analysis of the Model

Theorem 1: The closed set $D = \left\{(S,I,R) \in R_+^3 : N \leq \frac{b}{\mu_1}\right\}$ is positively- invariant with non-negative initial values in R_+^3

Proof: Consider the feasible region D as defined above, then the rate of change of the total population is given by;

$$\frac{dN}{dt} = b - \mu N \quad (5)$$

It follows that $\frac{dN}{dt} \leq b - \mu N$. Hence, if $N(0) \leq \frac{b}{\mu_1}$, then $N(t) \leq \frac{b}{\mu_1}$. Therefore, all solutions of the model with initial values in D remain in D for all time t > 0 and this implies that D is positively invariant.

For equilibrium point, we set

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0 \quad (6)$$

For disease free equilibrium

Hence the uninfected equilibrium $\varepsilon_0 = \left(\frac{b}{\mu_1}, 0,0\right)$

Then the infected equilibrium is

$\left(\frac{\alpha b+\mu_2+\theta}{(\beta+\mu_1\alpha)}\right), \frac{b\beta-\mu_1(\mu_2+\theta)}{(\beta+\mu_1\alpha)(\mu_2+\theta)}, \left(\frac{b\beta-\mu_1(\mu_2+\theta)}{(\beta+\mu_1\alpha)(\mu_2+\theta)}\right)\frac{\theta}{\mu_3}$

Uninfected equilibrium

$(S,I,R) = \left(\frac{b}{\mu_1}, 0,0\right)$

Then denote the infected equilibrium by $\varepsilon^* = (S_0, I_0, R_0)$

$$(S_0,I_0,R_0) = \left(\frac{\alpha b+\mu_2+\theta}{(\beta+\mu_1\alpha)}\right), \frac{R_0-1}{(\beta+\mu_1\alpha)(\mu_2+\theta)}, \left(\frac{R_0-1}{(\beta+\mu_1\alpha)(\mu_2+\theta)}\right)\frac{\lambda}{\mu_3} \quad (7)$$

Analysis of Basic Reproduction Number (R_0)

The basic reproduction number of the model (4) with the disease free equilibrium point $\varepsilon_0 = \left(\frac{b}{\mu_1}, 0,0\right)$ is given as $R_0 = \frac{\beta b}{\mu_1(\mu_2+\theta)}$. The threshold quantity R_0 is the basic reproduction number of the normalized model system (4). It is the average number of new secondary infections generated by a single infected individual in his or her infectious period. (Ajao et al., 2023, Adesola et al., 2024b).

Local Stability of Disease Free Equilibrium

The local stability of ε_0 shall be determined using Jacobian matrix model (4).

Let

$$\frac{dS}{dt} = f_1 = b - \frac{\beta SI}{1+\alpha I} - \mu_1 S$$
$$\frac{dI}{dt} = f_2 = \frac{\beta SI}{1+\alpha I} - (\mu_2+\theta)I$$
$$\frac{dR}{dt} = f_3 = \theta I - \mu_3 R$$

Before finding the characteristic equation, we will evaluate the Jacobian matrix at DFE=$\left(\frac{b}{\mu_1}, 0,0\right)$. The Jacobian matrix of the model is computed below;

$$J((\varepsilon_0) = \begin{pmatrix} -\mu_1 & -\frac{\beta b}{\mu_1} & 0 \\ 0 & \frac{\beta b}{\mu_1} - (\mu_2+\theta) & 0 \\ 0 & \theta & -\mu_3 \end{pmatrix} \quad (8)$$

The characteristic equation of (4) above are obtained as $|J_{\varepsilon_0} - \lambda I|$=0, where I is the (3*3) identity matrix.

Then,

$$|J\varepsilon_0 - \lambda I| = \det\begin{pmatrix} -\mu_1-\lambda & -\frac{\beta b}{\mu_1} & 0 \\ 0 & \frac{\beta b}{\mu_1} - (\mu_2+\theta) - \lambda & 0 \\ 0 & \theta & -\mu_3-\lambda \end{pmatrix} \quad (9)$$

$$(-\mu_1-\lambda)(-\mu_3-\lambda)\left(\frac{\beta b}{\mu_1} - (\mu_2+\theta) - \lambda\right)$$

The eigenvalues of the Jacobian matrix are; $\lambda = -\mu_1, \lambda = -\mu_3, \lambda = \frac{\beta b}{\mu_1} - (\mu_2+\theta)$

Because $\mu_2 > 0$, there are two cases for equilibrium behavior:

(1) If $\frac{\beta b}{\mu_1} - (\mu_2+\theta) < 0$, then DFE is a stable node

(2) If $\frac{\beta b}{\mu_1} - (\mu_2+\theta) > 0$, then DFE is a saddle point

Obviously, if $R_0 < 1$, then the eigen value $\frac{\beta b}{\mu_1} - (\mu_2+\theta)$ is negative, then $\varepsilon_0$is locally asymptotically stable, if $R_0 > 1$, then, the eigen value $\frac{\beta b}{\mu_1} - (\mu_2+\theta)$ is positive, hence, $\varepsilon_0$is unstable.

Theorem 2: If $R_0 \leq 1$, then the disease free equilibrium ε_0 of the system (4) is globally asymptotically stable

Proof: To establish the global stability of the disease free equilibriumε_0, we construct the following Lyapunov function $L: \Omega \to R$

$L(S, I) = I$

Calculating the derivative of Lalong the solution of the proposed system, we obtain

$$L^I = \frac{\beta SI}{1+\alpha I} - (\mu_2 + \theta)I$$

$$= (\mu_2 + \theta)\left(\frac{R_0 S}{1+\alpha I} - 1\right) I \qquad (10)$$

We see that

$L^I \leq 0$, for$R_0 < 1$.

If $R_0 < 1$ then$L^I = 0 \Leftrightarrow I = 0$.

If $R_0 = 1$ then$L^I = 0 \Leftrightarrow S = 1$.

Therefore by LaSalle's invariance principle (1987), the disease free equilibrium is locally asymptotically stable.

Theorem 3: If$R_0 > 1$, the endemic equilibrium ε^* of the system (4) is locally asymptotically stable if $c_3 > 0$and $c_1 c_2 - c_3 > 0$.

Proof: Linearizing the Jacobian matrix of system (4) of ε^*;

$$J(\varepsilon^*) = \begin{pmatrix} -\frac{\beta I^*}{1+\alpha I^*} - \mu_1 & -\frac{\beta S^*}{(1+\alpha I*)^2} & 0 \\ \frac{\beta I^*}{1+\alpha I^*} & \frac{\beta S^*}{(1+\alpha I*)^2} - (\mu_2 + \theta) & 0 \\ 0 & \theta & -\mu_3 \end{pmatrix} \qquad (11)$$

The characteristic equation of the matrix equation (11) is

$\lambda^3 + c_1\lambda^2 + c_2\lambda + c_3 = 0$

$c_1 = \mu_3 + B + \mu_2 + \theta + A + \mu_1$

$c_2 = A(\mu_3 + \mu_2 + \theta) + B(\mu_3 + \mu_1) +$

$\mu_1(\mu_3 + \mu_2 + \theta) + \mu_3(\mu_2 + \theta)$

$c_3 = \mu_3(A(\theta + \mu_2) + B\mu_1 + \mu_1(\mu_2 + \theta))$

Where $A = \frac{\beta I^*}{1+\alpha I^*}$and $B = \frac{\beta S^*}{(1+\alpha I^*)^2}$

According Hurwitz criterion, when $R_0 > 1$, the endemic equilibrium $\varepsilon^*$of system (4) is locally asymptotically stable if $c_1 > 0$and $c_1 c_2 - c_3 > 0$.

Theorem 4: (Dulac's Criterion)

Consider the following general nonlinear autonomous system

$x(t) = f(x), x \in E \qquad (12)$

Let $f = C^I(E)$ where E is a simple connected region inR^2. If there exists a function $H \in C^I(E)$ such that $\nabla.(H.f)$ is not identically zero and does not change sign in E, the system (12) has no close orbit lying entirely in E. if A is an annular region contained in E on which $\nabla.(H.f)$ does not change sign, then there is at most one limit cycle of the system (12) in A.

Theorem 5: (The Poincare-Bendixson Theorem): Suppose that $f \in C^I(E)$

Where E is an open subset of $R^n$and that the system (12) has a trajectory Γ contained in a compact subset $f$of E. Assume that the system (12) has only one unique equilibrium point $x_0$in f, then one of the following possibilities holds.

(1) $w(\Gamma)$ is the equilibrium point x

(2) $w(\Gamma)$ is a periodic orbit

(3) $w(\Gamma)$ is a graphic

Theorem 6: Let ε^* be the unique positive equilibrium point of the system (4), If$R_0 > 1$, then endemic equilibrium ε^* of the system (4) is globally asymptotically stable.

Proof: Using theorem 4 and 5, consider;

$H(S, I, R) = \frac{1}{SIR}, S > 0, I > 0$, and $R > 0$,

$\nabla.(H.f) =$

Then$\frac{\partial}{\partial S}(H.f_1) + \frac{\partial}{\partial I}(H.f_2) + \frac{\partial}{\partial R}(H.f_3) \qquad (13)$

$$= \frac{\partial}{\partial S}\left[\frac{1}{SIR}\left(b - \frac{\beta SI}{1+\alpha I} - \mu_1 S\right)\right] +$$

$$\frac{\partial}{\partial I}\left[\frac{1}{SIR}\left(\frac{\beta SI}{1+\alpha I} - (\mu_2 + \theta)I\right)\right] +$$

$$\frac{\partial}{\partial R}\left[\frac{1}{SIR}(\theta I - \mu_3 R)\right]$$

$$= \frac{-b}{S^2 IR} - \frac{\alpha\beta}{R(1+\alpha I)^2} - \frac{\theta}{SR^2} \qquad (14)$$

$$= -\left(\frac{b}{S^2 IR} + \frac{\alpha\beta}{R(1+\alpha I)^2} + \frac{\theta}{SR^2}\right) < 0 \qquad (15)$$

Hence, according to Dulac's criterion, there is closed orbit in the first quadrant; therefore, the endemic equilibrium is globally asymptotically stable.

Numerical Simulation

In this segment, we employ an iterative approach to determine the numerical simulation. When performing the numerical simulation, we take into account the parameters values presented in table 2. We apply the Runge-Kutta order (4) scheme to solve our model system (4) and the results are presented as follows;

RESULTS AND DISCUSSION

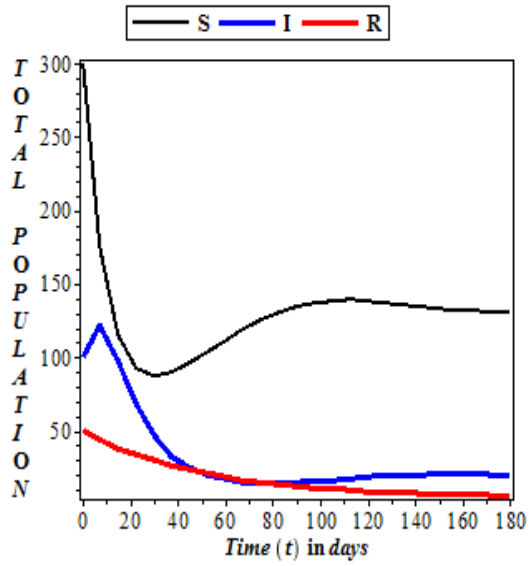

Figure 2:The Population of Susceptible, Infected and Recovered Individuals

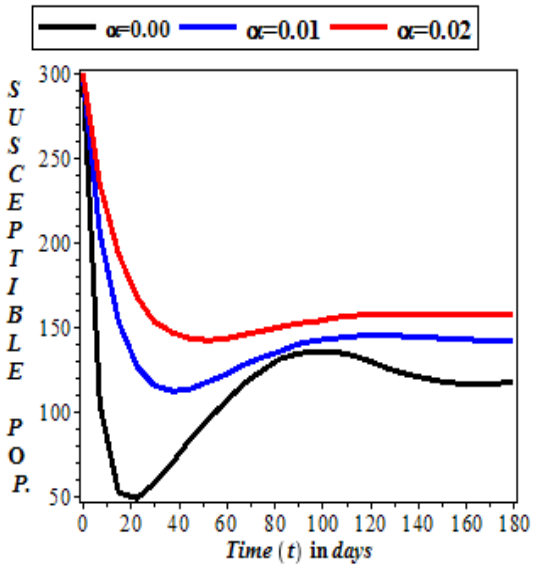

Figure 3:The Population of Susceptible, Individuals when $\alpha = 0.0,0.01\&0.02$

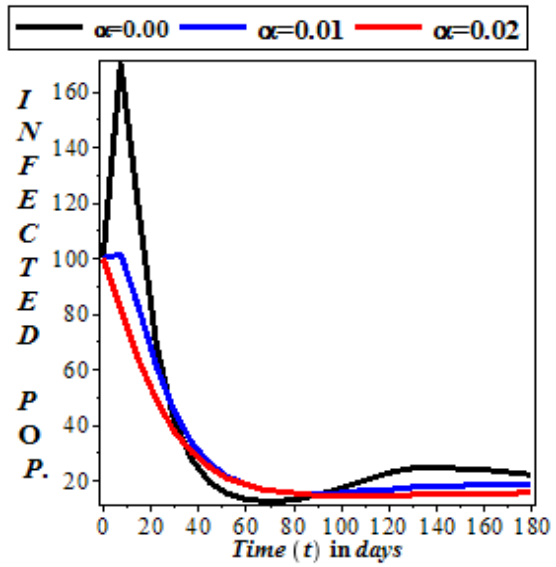

Figure 4:The Population of Infected Individuals when $\alpha = 0.0,0.01\&0.02$

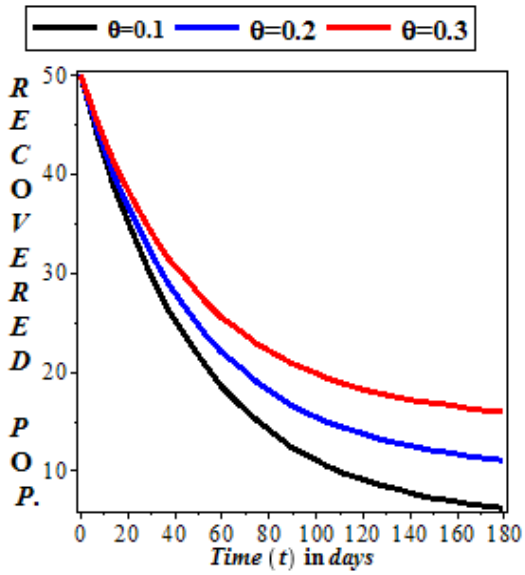

Figure 5:The Population of Recovered, Individuals when $\theta = 0.1,0.2\&0.3$

Figure 2 provides a comprehensive illustration of the dynamic interaction among susceptible, infected, and recovered individuals, capturing the nuanced changes in the populations of these three distinct groups over time. Within the SIR model framework, susceptibility transitions into infection when susceptible individuals come into contact with infected counterparts. The infection rate is intricately influenced by both the transmission rate and the prevailing number of infected individuals within the population.

Moreover, infected individuals within the model have two potential outcomes: recovery or mortality. The recovery rate emerges as a critical determinant, shaping the trajectory of the epidemic. Those who successfully recover from the infection gain immunity, effectively transitioning into the recovered category. Importantly, recovered individuals no longer contribute to the spread of the disease, serving as a key element in breaking the chain of transmission within the population. This intricate interplay of susceptibility, infection, recovery, and immunity encapsulates the fundamental dynamics of the SIR model.

In Figure 3, the susceptible population is analyzed across various memory term values, specifically, $\alpha =$

0.0,0.01&0.02. These values represent different degrees of memory incorporation, reflecting the influence of past and current information on the disease's status. The findings highlight a notable trend: as the memory term increases substantially, there is a corresponding rise in the susceptible population. This observed increase in susceptibility implies a prolonged duration during which individuals remain susceptible before transitioning into the infected state. The larger memory term, indicative of enhanced information retention, appears to extend the vulnerability of individuals to infection, shaping the dynamics of disease transmission. The insights derived from Figure 3 underscore the pivotal role of memory in influencing the susceptibility dynamics within the context of the epidemic model.

In Figure 4, the impact of the memory term on the dynamic spread of the disease is illustrated. As the memory term decreases, specifically to lower values, the population of infected individuals exhibits an exponential increase. This suggests that reduced memory, representing limited information retention, is associated with a more rapid and sustained rise in the number of infected individuals. Conversely, as the memory term increases to$\alpha = 0.01\&0.02$, there is a noticeable dampening effect on the dynamic spread of the disease. The population of infected individuals starts to decline, indicating that a higher degree of memory incorporation contributes to a mitigated spread of the disease over time. This observation emphasizes the significance of information retention, as reflected by the memory term, in influencing the trajectory of the epidemic and its potential to curb the rise in infections.

In Figure 5, the population of recovered individuals is depicted, showcasing the impact of varying recovery rates0.1,0.2&0.3. As the recovery rate increases, there is a noticeable growth in the number of individuals transitioning to the recovered state. This observation highlights the direct relationship between the recovery rate and the pace at which individuals recuperate from the infection. Specifically, a higher recovery rate corresponds to an accelerated recovery process, leading to an increased population of individuals who have successfully overcome the infection and entered the recovered category. This insight underscores the crucial role of the recovery rate in influencing the dynamics of the epidemic, with higher recovery rates contributing to a more substantial pool of recovered individuals over time.

CONCLUSION

In this present study, our investigation highlights the pivotal role of a memory term in shaping the dynamic control of epidemic diseases, as evidenced by the insightful data presented in Figures 1 to 4 of our SIR epidemic model. The results distinctly indicate that a substantial memory term, indicative of ample information, plays a critical role in empowering medical practitioners to devise more effective and targeted control strategies for managing and mitigating the spread of the disease. This underscores the significance of considering the impact of memory and information in the intricate process of modeling and controlling epidemics. In essence, our findings emphasize the need for a nuanced understanding of the interplay between memory, information, and epidemic dynamics to foster more resilient and responsive public health interventions.

REFERENCES

Adeniran G. A., Olopade I. A., Ajao S. O., Akinrinmade V. A., Aderele O. R., & Adewale S. O. (2022). Sensitivity and Mathematical Analysis of Malaria and Cholera Co-Infection. *Asian Journal of Pure and Applied Mathematics*, *4*(1), 425–452.

Adesanya A. O., Olopade I. A., Akinwumi T. O. & Adesanya A. A. (2016). Mathematical Analysis of Early Treatment of Gonorrhea Infection. *American International Journal of Research in Science, Technology, Engineering & Mathematics*, 15(2).

Adesola O. I., Oloruntoyin S. S., Emmanuel P. M., Temilade M. I., Adeyemi A. G., Oladele A. S., Mamman A. U., & Kareem A. A. (2024). Mathematical Modelling and Analyzing the Dynamics of Condom Efficacy and Compliance in the Spread of HIV/AIDS. *Asian Research Journal of Current Science*. 6(1): 54–65.

Adesola O. I., Temilade M. I., Emmanuel P. M., Oladele A. S., Adeyemi A. G., Sunday S, & Olumuyiwa A. S. (2024). Mathematical Analysis of Optimal Control of Human Immunodeficiency Virus (HIV) Co-infection with Tuberculosis (TB). *Asian Research Journal of Current Science*. 6(1), 23–53.

Adewale S. O., Olopade I. A., Ajao S. O., & Adeniran G. A. (2015a). Mathematical Analysis of Diarrhea in the Presence of Vaccine. *International Journal of Scientific and Engineering Research*. 6: 396-404.

Adewale S. O., Olopade I. A. & Adeniran G. A. (2015b). Mathematical Analysis of Effects of Isolation on Ebola Transmission Dynamics. *Researchjouranli's Journal of Mathematics*. 2: 1-21.

Ajao S., Olopade I., Akinwumi T., & Adesanya O. (2023). Understanding the Transmission Dynamics and Control of HIV Infection: A Mathematical Model Approach. *Journal of the Nigerian Society of Physical Sciences.* 5(2): 1389. DOI:https://doi.org/10.46481/jnsps.2023.1389.

Akinwumi T. O., Olopade I. A., Adesanya A. O. & Alabi M. O. (2021). A Mathematical Model for the

Transmission of HIV/AIDS with Early Treatment. *Journal of Advances in Mathematics and Computer Science*; 36(5): 35-51.

Almuqrin M. A., Goswami P. & Sharma S. (2021). Fractional model of Ebola virus in population of bats in frame of Atangana- Baleanu fractional derivative," *Results in Physics.* 26: 104295.

Alzaid S. S., Alkahtani B. S. T. & Sharma S. (2021). Numerical solution of fractional model of HIV-1 infection in framework of different fractional derivatives," *Journal of Function Spaces*. 10: 20-21.

Beretta E. & Takeuchi Y. (2010). The class of discrete SIR epidemic models with distributed delay. *Nonlinear Anal*ysis. 28:1909–1921.

Brauer F. & Castillo-Chávez C. (2001). Mathematical Models in Population Biology and Epidemiology, *Springer-Verlag*, New York.

Capasso V. (2008). Mathematical Structures of Epidemic Systems, *Springer Verlag*, Berlin, 2nd edition.

Cooke K. L. (1979). Stability analysis for a vector disease model, *Rocky Mountain J. Math*. 9: 31–42.

Diekmann O., Heesterbeek H. & Britton T. (2013). Mathematical Tools for Understanding Infectious Disease Dynamics. *Princeton University Press*. Princeton.

Foy B. H., Wahl B., & Mehta K. (2021). Comparing COVID-19 vaccine allocation strategies in India: a mathematical modelling study. *International Journal of Infectious Diseases*. 103: 431–438.

Ibrahim M. O., Akinyemi S. T. & Dago M. M. (2015). Mathematical Modelling of a Staged Progression HIV/AIDS Model with Control Measures. *Journal of Nigerian Association of Mathematical Physics.* 29: 163-166.

Kermack W. O. & McKendrick A. G. (1927). A contribution to the mathematical theory of epidemics," *Proceedings of the Royal Society of London*. 115:700–721.

LaSalle J. P. (1987). The stability of dynamical systems. *Society for Industrial and Applied Mathematics*. 25.

Olopade I. A., Adesanya A. O. & Mohammed I. T. (2017). Mathematical Analysis of the Global Dynamics of an SVEIR Epidemic Model with Herd Immunity. *International Journal of Science and Engineering Investigations. (IJSEI)*. 6(69) :141-148.

Olopade I. A., Adesanya A. O. & Akinwumi T. O. (2021a). Mathematical Transmission of SEIR Epidemic Model with Natural Immunity. *Asian Journal of Pure and Applied Mathematics*. 3(1) :19-29.

Olopade I. A., Adewale S. O., Mohammed I. T., Adeniran G. A., Ajao S. O. & Ogunsola A. W. (2021b). Effect of Effective Contact Tracing in Curtaining the Spread of Covid-19. *Asian Journal of Research in Biosciences. 3*(2): 118-134.

Olopade I. A., Ajao S. O., Adeniran G. A., Adamu A. K., Adewale S. O., & Aderele O. R. (2022). Mathematical Transmission of Tuberculosis (TB) with Detection of Infected Undetectected. *Asian Journal of Research in Medicine and Medical Sciences*. *4*(1): 100-119.

Olopade I. A., Akinola E. I., Philemon M. E., Mohammed I. T., Ajao S. O., Sangoniyi S. O., Adeniran G. A. (2024c). Modeling the Mathematical Transmission of a Pneumonia Epidemic Model with Awareness. *J. Appl. Sci. Environ. Manage.* 28(2): 403-413.

Philemon M. E., Olopade I. A., & Ogbaji E. O. (2023). Mathematical Analysis of the Effect of Quarantine on the Dynamical Transmission of Monkey-Pox. *Asian Journal of Pure and Applied Mathematics*. 5(1): 473–492.

Ramos A. M., Ferrández M. R., & Vela-Pérez M. (2021). A simple but complex enough θ-SIR type model to be used with COVID-19 real data. Application to the case of Italy. *Physica D*. 421, article 132839.

Rao F., Mandal P. S., & Kang Y. (2019). Complicated endemics of an SIRS model with a generalized incidence under preventive vaccination and treatment controls. *Applied Mathematical Modelling*. 67: 38–61.

Safiel R., Massawe E. S., & Makinde O. D. (2012). Modelling the Effect of Screening and Treatment on Transmission of HIV/AIDS Infection in a Population. *American Journal of Mathematics and Statistics*. 2(4): 75-88. DOI: 10.5923/j.ajms.20120204.03.

Sajid M., Abbas Z., Ali N., & Javed T. A. (2013). Note on Solutions of the SIR Models of Epidemics Using Ham. *ISRN Applied Mathematics*; Article ID 457072, 4 pages.

Srivastava H. M., Shanker D. R., & Jain M. (2019). A study of the fractional-order mathematical model of diabetes and its resulting complications. *Mathematical Methods in Applied Sciences* 42